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THE COURSE OF EVENTS IN SECONDARY WOUND SHOCK *

W. B. CANNON, M.D.
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In the issues of THE JOURNAL of February 23 and March 2, 1918, there was published a series of articles by Cowell, Fraser, Hooper and myself which described certain clinical observations that we had made on soldiers who suffered from shock and allied conditions. These observations confirmed earlier reports on the persistent low arterial pressure, the rapid pulse and respiration, and the lowered body temperature of the shock state; they also revealed a concentration of corpuscles in the capillaries, a reduction of the alkali reserve corresponding in general to the degree of lowering of arterial pressure, a marked sensitiveness to ether or chloroform anesthesia, and a tolerance of nitrous oxid and oxygen as an anesthetic. On the basis of these facts a definition of traumatic or wound shock might be offered; it is a general bodily state occurring after severe injury and characterized by persistent low arterial pressure, rapid pulse, pallor or slight cyanosis, sweating, superficial rapid respiration, and usually by a dulled mental condition.

EXEMIA AS A CAUSE OF LOW PRESSURE

Among the articles in the series above mentioned was a theoretical discussion of the nature of wound shock, in which the low blood pressure was regarded as the central fact of the fully developed complex.[1] Reasons were given for refusing acceptance of the

* Read before the Section on Surgery, General and Abdominal, at the Seventieth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1919.

1. Cannon, W. B.: A Consideration of the Nature of Wound Shock, J. A. M. A. **70**: 611 (March 2) 1918.

acapnia theory and the nerve exhaustion theory of the low pressure. The factors determining arterial tension were then analyzed, with the result that the low pressure was ascribed to "exemia"; that is, a temporary lessening of the volume of circulating blood, though not a loss of blood from the body. The possibility that the acidosis of shock, if carried to a sufficient degree, might play a rôle in causing further exemia was considered, but the occasion for the primary fall of pressure was left unsettled.

Shortly after the foregoing articles were presented as a report to the Subcommittee on Shock of the English Medical Research Committee, opportunity was offered at University College, London, to cooperate with Prof. W. M. Bayliss in an experimental analysis of the phenomena which had been observed clinically. Unfortunately, that work was cut short by military necessities, and the possibility of resuming it did not arise until the establishment of the Laboratory of Surgical Research of the American Expeditionary Forces at Dijon, France. Much that appears later in this report has come from observations made in London and from observations made by the group working at Dijon.[2]

PROPER EVALUATION OF THE DIFFERENT AGENTS IN SHOCK

That the phenomena of shock require experimental analysis is clear from a consideration both of its inherent complexity and of its attendant conditions. Shock has long been one of the great mysteries in surgery, and because of associated hemorrhage and infection there is usually much difficulty in assigning proper values to the different agents which might produce the general state of depression. No doubt hemorrhage may have an important part in the development of this state, and infection likewise is important; but there are recorded cases in which both hemorrhage and infection were absent or slight and in which, never-

2. Already reports of these observations have appeared: Cannon: Med. Bull. Med. Research Society of the American Red Cross, Paris, April, 1918, p. 426; Compt. rend. Soc. de biol. **81**: 850 (Oct. 19) 1918. Bayliss: Intravenous Injection in Wound Shock, London, 1918. Cannon and Bayliss: Note on Muscle Injury in Relation to Shock, Report of Special Committee on Surgical Shock, English Medical Research Committee, March 14, 1919, p. 19. Cannon: Some Characteristics of Shock Induced by Tissue Injury, ibid., p. 27; Mil. Surgeon **44**: 494 (May) 1918.

theless, shock was observed. These facts of observation have led to the view that, beside hemorrhage and infection, there is an unknown factor at work.

Various views have been advanced to account for the nature and operation of this unknown factor. It was early suggested that serious wounds exhaust the nerve centers controlling the blood flow and that in consequence arterial pressure falls. This view has been extended so as to include the idea that the shocked man is suffering from general exhaustion not only of nerve cells, but of other parts of the body as well. Another view which has been widely noticed is that excessive respiration, by reducing the carbon dioxid content of the blood, causes such vascular changes as to reduce the volume of circulating blood. And still another suggestion is that the low pressure follows accumulation of fat either in the lungs or in the nerve centers governing the circulation; in the former instance the low pressure would be due to mechanical obstruction in the pulmonary capillaries, and in the latter case it would be due to injury of the vasoconstrictor center by anemia. None of these views as to the unknown factor in shock has been generally accepted. Possibly, as will be seen later, certain of them may at some stage have significance in the production of shock. The fact that the condition of shock has long remained unexplained, however, indicated the need for further investigation. The question to be settled seemed to be a determination of what factor, naturally related to the onset of shock, may so operate in the body, when hemorrhage and infection are ruled out, as to induce the characteristic persistent low blood pressure of the shock state.

DUPLICATING IN EXPERIMENTS THE CONDITIONS SURROUNDING SHOCK

One of the fundamental principles of experimental research is that the phenomenon to be studied shall present, when experimentally reproduced, as much detailed similarity as possible to the phenomenon as it occurs naturally. This is a principle which has been largely neglected in experimental investigations of shock. There is no doubt that a persistent low blood pressure can be produced by a variety of means. Investigators have reported the duplication of certain

aspects of shock by prolonged stimulation of nerves, by severe and long-continued manipulation of exposed intestines, by the application of acid to the abdominal viscera, by vigorous over-ventilation of the lungs, by repeated punching of the diaphragm, or by injecting into the circulation large amounts of cream. It is possible that such procedures induce changes similar to those which may occur naturally in shock; but no one can successfully argue that such procedures reproduce the circumstances which occasion shock after ordinary wounds or injury. Obviously, it is desirable to duplicate so far as possible the actual conditions which give rise to shock and then to analyze them to determine what factor among them is operative.

Shock occurs in warfare on an enormous scale. To find evidence of the interest which the subject of shock has aroused at times of fierce strife one needs only to look through any large catalogue of medical literature and observe the dates of publication of articles. In warfare the shock state occurs characteristically in association with extensive and severe wounds—wounds in which large tissue masses are shredded, torn or crushed; as, for example, when there has been rupture and mashing of muscle by shell fragments. Similarly in civil life, traumatic shock is seen characteristically after injuries under car wheels or in machinery, when limbs have been badly mangled.

As Cowell[3] pointed out, there are certain very severe wounds in which death is certain to occur soon because of the anatomic damage which has been done. The patient's condition may be seriously complicated by abundant hemorrhage. In such cases, symptoms of shock are seen immediately; and from the earliest moment there is low pressure. This may be called "primary wound shock." On the other hand, there is testimony from a variety of sources[4] that after large wounds the state of shock commonly does not appear at once, but develops in the course of a few hours. This is Cowell's "secondary wound shock." When shock thus develops it may be attended by little hemor-

3. Cowell, E. M.: The Initiation of Wound Shock, J. A. M. A. **70**: 607 (March 2) 1918.

4. Archibald and McLean: Ann. Surg. **66**: 280 (Sept.) 1917. Sant: Bull. Mém. Soc. de Chir. **44**: 207 (Feb. 6) 1918. Quénu: Presse méd. **26**: 69 (Feb. 7) 1918.

rhage, and it may occur too soon to be due to infection. Since these facts have been actually observed by medical men and dressers working in front line relief stations, there is justification for looking for the development of a state of shock in which hemorrhage and infection are negligible factors.

EXPERIMENTAL PRODUCTION OF SHOCK

In order to duplicate in lower animals conditions which give rise to shock in man, the thigh muscles in the anesthetized cat were traumatized by means of a blunt wedge-shaped hammer. While being struck, the muscles were supported by an iron block. The trauma usually failed to break the skin, so that infection

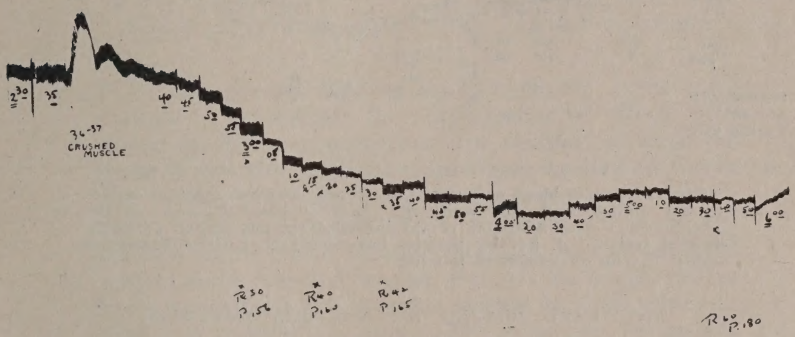

Fig. 1.—Typical fall of arterial blood pressure after crushing muscles of the thigh; *R*, respiration; *P*, pulse.

from without was impossible. Occasionally the femur was broken, but this had no important influence on the results which were seen. After this procedure the course of events was followed and the arterial pressure was recorded by means of a mercury manometer. In Figure 1 is presented a record of the blood pressure changes typically seen after such trauma.

As Figure 1 indicates, the crushing of the muscles in the hind leg may have no immediate effect. After about twenty minutes, however, a fall of arterial pressure begins, and after about an hour the pressure has usually fallen to 80 or 90 mm. of mercury or even lower; that is, to a shock level. Pressure may persist at this level for several hours. Obviously, a general

bodily condition resembling shock has been produced by duplication of circumstances which induce shock in man. The only variant from human conditions is the presence of anesthesia. That this is not a determining factor, however, is indicated by the use of a controllable anesthetic such as ether, which may be lightened as the animal goes into deeper and deeper stupor. The lessening of the ether concentration under these circumstances has no beneficial effect in improving the circulation.

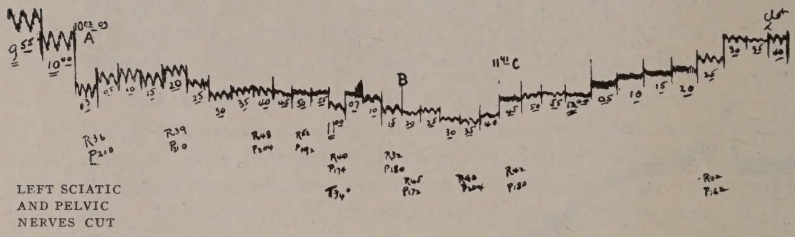

Fig. 2.—Fall of blood pressure after muscle injury though the nerves to the injured limb had been cut. Rise of blood pressure to the original level after tying (at 11:41) the blood vessels to and from the injured region. *A*, left leg muscles hammered; *B*, muscles hammered again; *C*, clip on vessels to left leg.

THE PART PLAYED BY NERVOUS IMPULSES

The general bodily state which has been caused by the crushing of tissues in one hind leg naturally raises the question as to whether the widespread effect in the organism is due to nervous impulses roused by the trauma. These impulses on passing to the central nervous system might so affect it as to produce profound depression of nerve cells and the exhaustion which is apparent both in experimental animals and in human beings when shocked. Is that the case? The nature of the experiment permits the settlement of this question. It is only necessary to transect the spinal cord above the lumbar plexus or to sever all nerves of the limb which is to be injured in order to disconnect the region from the central nervous system. When this has been done and the denervated muscles are traumatized, events occur similar to those seen after trauma with the nerves intact (Fig. 2). There is common testimony from men who have been seri-

ously injured that the wounds are not painful; in other words, afferent impulses are not arriving from the injured region in any such intensity as to affect deeply the central nervous system. It would be rash, however, to argue that nervous impulses play no part in the establishment of shock; indeed, there are cases in which a pure nervous shock from trivial wounds seems to be present. From personal experience and from testimony of other observers, however, I am justified in reporting that these cases are rare. The vast majority of shock cases are associated with severe tissue damage. As shown in Figure 2, there is no essential relation between the production of shock in such instances and excessive stimulation of the central nervous system.

TOXIC MATERIAL PROCEEDING FROM WOUNDED TISSUES

If the general bodily state resulting from local trauma is not mainly the consequence of effects produced on the nervous system, the relation between the general and the local conditions may be looked for in the other great integrating system, namely, the circulatory. Quite possibly there is given off, from the injured tissue to the flowing blood or lymph, material which, when carried to the rest of the body, proves toxic and affects the vascular system in such a way as to lower arterial pressure. The idea can be readily tested. The blood vessels of the leg (the iliac artery and vein) are tied and the muscle then crushed. As stated above (Fig. 1), the arterial pressure usually begins to fall in the cat about twenty minutes after the injury. In Figure 3, the blood vessels of the leg were tied before the muscles were smashed, and ligatures were left in place for thirty-three minutes after the injury. The record shows that there was no dropping of blood pressure during this period; but as soon as the blood flow was restored, the pressure promptly fell to a low level.

The fall of pressure after restoration of blood flow in the traumatized limb might be regarded as due to loss of blood and lymph into the damaged tissues and not to the washing out of toxic material from these tissues into the rest of the body. This suggestion was put to test by removing with symmetrical cuts the two hind

legs and weighing them. The difference in weight, which in some instances was only 10 per cent. of the estimated blood volume, would not represent enough extravasated blood to account for the fall of pressure. The conclusion appears justified, therefore, that a pressure-lowering substance passes from the traumatized region to the rest of the body by way of the circulation. Extravasation of blood may augment the depressant effect.

The conclusion just stated receives support from an experiment in which the muscle was injured, and while the pressure in consequence was falling, the vessels of the leg (the iliac artery and vein) were clipped. As shown in Figure 2, the occlusion of the vessels was

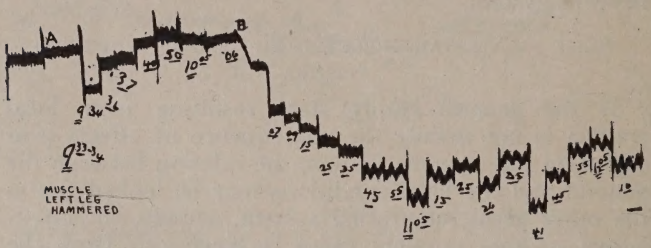

Fig. 3.—Failure of blood pressure to fall at the usual time after muscle injury if the blood vessels to the injured region have been tied. On restoration of the blood flow, the pressure promptly falls. *A*, vessels clamped; *B*, clamps off.

followed by a progressive rise of pressure to the normal level. This evidence indicates, therefore, that whatever may be the substance arising from the injured tissues, it is fairly promptly changed in the body so that its effect is not permanent.

Experiments which have been performed by Cattell since returning to the United States indicate that the fall of blood pressure is not, at least in the early stages, associated with dilatation of the arterioles. This evidence is in agreement with that reported by Seelig and Lyon,[5] by Mann,[6] and also by Erlanger and Gasser.[7]

5. Seelig and Lyon: Surg., Gynec. & Obst. **11**: 146 (July) 1910.
6. Mann: Bull. Johns Hopkins Hosp. **25**: 208 (July) 1914.
7. Erlanger and Gasser: Ann. Surg. **69**: 391 (April) 1919.

It is further testimony against the view that shock is primarily due to nervous exhaustion.

Careful examination was made of the lungs in several of these cases by Col. L. B. Wilson and by Major Henry W. Cattell, and in no instance was there evidence of any accumulation of fat. Furthermore, there was no twitching or other disturbance which might indicate that fat had passed through the pulmonary capillaries and into the central nervous system. The fall of pressure, therefore, cannot be attributed to fat embolism.

Although there was usually a change of respiration in the direction of greater rapidity and lessened amplitude, this was not such as to cause a washing out of carbon dioxid and a consequent acapnia. The lowered blood pressure, therefore, cannot be ascribed to such a change in the blood.

For a long time it has been known that tissue extracts, when injected into the blood stream, will cause a temporary fall of pressure. In 1903, Vincent and Sheen[8] proved that watery extracts of a variety of tissues would produce this effect. Furthermore, Dale and Laidlaw have shown that a characteristic shock-like condition can be induced by the injection of extremely minute amounts of histamin, a substance which Barger and Dale proved present in the mucosa of the small intestine.[9] The low pressure caused by this substance is not due to dilatation of the arterioles but apparently to dilatation of the capillaries with escape of plasma into the tissue spaces.[10] This effect is in consonance with the evidence for diminished volume of circulating fluid and concentration of the corpuscles in capillaries in human cases. French surgeons with extensive experience in the war, Delbet[11] and Quénu,[12] have independently come to the conclusion that the phenomena of secondary shock are the consequence of absorption of proteolytic products arising

8. Vincent and Sheen: J. Physiol. **29**: 264 (April) 1903.
9. Dale and Laidlaw: J. Physiol. **41**: 318 (Dec.) 1910 and 499 (Jan.) 1911; **43**: 182 (Oct.) 1911; **52**: 355 (March) 1919.
10. Dale and Richards: J. Physiol. **52**: 110 (July) 1918.
11. Delbet: Bull. de l'Acad. de méd., Paris **80**: 13 (July) 1918.
12. Quénu: Bull. et mém. Soc. de chir. de Paris **44**: 496, 1918; Presse méd. **26** (Feb. 7) 1918.

from the region of the injury; and Turck[13] has expressed similar ideas. There is the possibility that this *traumatic toxemia* may be closely related to "peptone" shock and that the toxic agent is, like "peptone," capable of making the capillary wall more permeable to the fluid portion of the blood. These suggestions must be regarded, however, as purely hypothetic. It is clear that a large field for investigation is opened by the facts above described.

A SECONDARY DAMAGE

Besides the primary fall of blood pressure induced by substances given off by the injured tissues, there is a secondary damage, which may result from the low blood pressure itself. As stated above, there com-

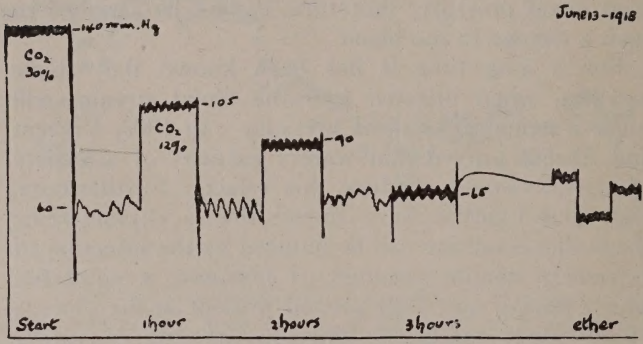

Fig. 4.—Record of an experiment showing the progressively harmful effects on the circulation of persistent low arterial pressure.

monly appears in clinical cases of shock a reduction of the alkali reserve of the blood, or an "acidosis" in the sense defined by J. L. Henderson. The reduction of the reserve corresponds roughly to the degree of lowering of blood pressure. In forty-four cases of shock and hemorrhage which I investigated at Béthune in 1917, the relations between blood pressure and the alkali reserve were as shown in the accompanying table.

When we consider that 50 per cent. by volume of carbon dioxid capacity is close to the boundary between normality and acidosis, it is obvious that the main-

13. Turck: M. Rec. **89**: (June) 1918; **90**: 471 (March) 1919.

tenance of a blood pressure above 80 mm. causes little alteration in the blood, but that a reduction of the pressure below this level results in a marked diminution of the sodium bicarbonate as indicated by the carbon dioxid given off. These clinical observations have been confirmed by experiments. A cannula introduced through the chest wall and tied into the pericardium permits fluid to be introduced into the pericardial sac. By varying the pressure of this fluid, the output from the heart can be varied or held at any given level. When this is done, the interesting result is observed that unless there has been considerable hemorrhage, a blood pressure in the neighborhood of 80 mm. of mercury results in no diminution of the alkali reserve. On the other hand, a reduction of the pressure to 70, 60 or 50 mm. will cause, at the end of half an hour or an hour, a marked reduction of this reserve (Fig. 4), varying directly with the degree of lowering of the blood pressure. There appears to be, consequently, a critical level in the blood pressure below which it may not fall without bringing about a change in the character of the blood itself.

RELATION BETWEEN BLOOD PRESSURE AND ALKALI RESERVE

Systolic Blood Pressure, Mm.	Average Carbon Dioxid Capacity Vols. %
Above 80	49
70-80	43
60-70	35
Below 60	27

In the theoretical article previously referred to the suggestion was offered that the acidosis observed in shock may, when it becomes sufficiently developed, take part in a vicious circle of causation which might lead to still further reduction of blood pressure. This idea was put to experimental test, and it was found that if a volatile anesthetic, such as ether, is employed, the alkali reserve can be very greatly reduced by injection of acid without any noteworthy fall of arterial pressure.[14]

14. Acidosis and Shock, Report No. 9 of Special Committee on Shock and Allied Conditions, English Medical Research Committee, October, 1918.

From these observations it appears clear that the reduction of the alkali reserve in the flowing blood has in itself no serious effect in the direction of augmenting the shock state.[15]

REDUCTION OF THE ALKALI RESERVE

The reduction of the alkali reserve may be accounted for on the basis of retarded blood flow. When the head of arterial pressure is lowered, the blood flow becomes slower. Observations by Gesell have revealed that a fall of arterial pressure from the normal to a low shock level (59 mm. of mercury) may be accompanied by the reduction of the flow to one fifth of its former speed.[16] Under such circumstances the number of trips which the corpuscles make between the lungs and the tissues will be reduced approximately to the same extent; thus, in such a case, though every corpuscle may leave the lungs fully laden with oxygen, the total amount of oxygen delivered to the tissues in unit time may be lowered, other things being equal, to one-fifth the usual amount.

It has been known for many years, chiefly from the work of Araki,[17] that any condition which diminishes the oxygen supply of the tissues, such as rebreathing expired air, or extreme slowing of respiration by morphin or by hemorrhage or poisoning with carbon monoxid, will lead to the production of lactic acid rather than carbonic acid as a tissue metabolite. Since lactic acid is nonvolatile, it will not be breathed away as is the case with carbonic acid. It will unite with the sodium of the sodium bicarbonate, forming sodium

15. In a discussion of theories of shock, Meltzer (Penn. M. J. **22**: 129 [Dec.] 1918) has stated that I became "converted to the theory that the most essential factor in the production of shock is acidosis." I have never published the idea that there was in the acidosis of shock a primary cause for the low pressure. At one time I did advocate the use of sodium bicarbonate as a means of avoiding the extreme acidosis which may follow surgical operation on shocked men. Men who were in great distress from air hunger after operation were made immediately comfortable by alkaline therapy. Because of this effect the suggestion was offered that the injection be made during the period when the pressure is likely to fall; that is, during operation. Thus the pressure was held up and the alkaline content of the blood increased. At that time it seemed possible that anesthesia and operation might in themselves produce a harmful acidosis. Later experimental work proved that the acidosis was probably purely secondary to the fall of pressure and that the main object should be to avoid the fall. Alkaline therapy thus became limited to the relief of symptoms of extreme acidosis. Because alkaline therapy was suggested is no warrant for assuming the conviction that shock is primarily due to acidosis.

16. Gesell: Am. J. Physiol. **47**: 491 (Jan.) 1919.

17. Araki: Ztschr. f. physiol. Chem. **15**: 335, 1891.

lactate, thus diminishing the alkali reserve. The carbon dioxid which is set free will, of course, be promptly eliminated.

DANGER FROM INSUFFICIENT OXYGEN SUPPLY

On the basis of the foregoing evidence, the acidosis which appears as the blood pressure is lowered, though not dangerous in itself, is an indication of a really serious danger, namely, the danger arising from insufficient oxygen supply.

The tissues of the body are known to vary greatly in their sensitiveness to oxygen want. Some muscles will endure total anemia for at least three hours without undergoing destructive alterations. On the other hand, certain nerve cells of the cerebral cortex cannot endure total anemia for more than seven or eight minutes without suffering changes so profound as to be irreversible. There is especial danger, consequently, that the nervous system will be damaged by persistence of the blood pressure below the critical level.

As shown in Figure 4, the blood pressure may be lowered to 60 mm. of mercury for an hour, and at the end of that time, if the pressure is allowed to rise, it will be nearly restored to its normal level. If now the pressure is again lowered to 60 mm. and held there for an hour, it does not rise so high; and when held to this low level for another hour, it may not rise at all. Examination of the blood under these circumstances indicates that there has been no material escape of fluid from the vessels. The failure of the pressure to rise after persistence at the low level is to be regarded, therefore, as dependent on the damage done to control of the circulation by the inadequacy of the circulation itself.

The facts just developed offer a basis for the similarity which has been repeatedly noted between the phenomena of shock and the phenomena of hemorrhage. After the blood pressure has once been lowered in shock, the effects on the organism are in all respects similar to those which are produced by a marked loss of blood. The dictum of the older surgeons that "shock is hemorrhage and hemorrhage is shock" is thus justified. The significance of this similarity in the treatment of both shock and hemorrhage will appear later.

SECONDARY SHOCK

A review of the evidence presented above permits us to follow the course of events in secondary shock. It may be thus summarized: Toxic material given off by smashed and dying tissue affects the circulation in such a way as to cause a lowering of the blood pressure; the low pressure involves a slowing of the circulation; the slow circulation involves damage because of diminished oxygen supply; and in consequence highly sensitive structures, especially nerve centers, become injured and lose their tonic activity. Thus, though in the early stages of shock the low blood pressure may be associated with constriction of the arterioles, in the late stages the arterioles may be relaxed because of such damage to the vasoconstrictor center that it no longer holds the vessels in tonic contraction.

TREATMENT OF SHOCK

The principles of the treatment of shock are suggested by a consideration of the facts and inferences outlined above.

In the first place, if a part of the body has become shattered and useless through injury so that it is impossible to save it, the harmful toxic effect of the torn tissue on the rest of the body may be avoided by applying tightly, as near as possible to the injured region, a tourniquet. Extreme care should be taken *not to remove the tourniquet before operating at a point proximal to it.* The reason for this urgency is that when tissues are deprived of their blood supply they undergo at a slower rate the same changes which they undergo when mechanically traumatized. During the course of military operations last year, several cases were reported of the sudden production of shock on removal of a tourniquet which had been applied to stop bleeding and left in place for an indefinite period. These observations obviously support the view of shock which has been outlined above. If a tourniquet used to stop bleeding is to be left in place for a long period, it should be applied at the most distal effective location. The surgeon must be guided in his decision to save or sacrifice the part by questions of viability, of gross infection in the excluded region, and of danger to the rest of the body from its retention. The danger of the tourniquet should lead, whenever possible, to the

substitution of other methods of controlling hemorrhage. If the injured tissue can be removed promptly either by amputation or débridement, that should be done in preference to the application of a tourniquet.

Observations by Lieutenant Aub in the Surgical Research Laboratory of the American Expeditionary Forces at Dijon proved that there is a marked diminution of the heat production as the blood pressure is lowered. Furthermore, the man in shock sweats and thus loses heat both by evaporation and by greater conduction of heat through wet clothing, especially when he is exposed in cold weather. These considerations give a rational basis for the treatment of shock by warmth, a procedure universally recognized as of great value. Every effort should be made to avoid loss of heat from the shocked man or to restore heat that has already been lost. These objects may be attained by avoiding unnecessary exposure of the body and by use of hot drinks, warm water bottles, and proper blanketing.

The facts regarding the damaging effects of low arterial pressure which have been emphasized above should be emphasized again. If the pressure is allowed to remain below the critical level for a sufficient length of time, the vasoconstrictor center may be so profoundly affected that no measure will restore it to its normal condition. The transfusion of blood, for example, then results in only a temporary rise of arterial pressure, just such a rise as may be seen after total destruction of the center. As shown in Figure 4, the time element in the damaging effect of low pressure is of the utmost importance. If simple measures, such as warmth and fluids by mouth, do not in half an hour raise the pressure above the critical level of 80 or 90 mm. of mercury, the case should be treated as if it were a case of hemorrhage; and the treatment should be *prompt* in order to avoid the progressive damage as time passes. There appears to be little doubt that the best method for raising the blood pressure is transfusion of properly matched blood; by such means not only is the pressure raised but oxygen carriers are added to the circulation. If blood is not available, Bayliss' gum salt solution,[18] made of selected pure gum

18. Bayliss: Intravenous Injections in Wound Shock, London, 1918.

in physiologic sodium chlorid solution, or Erlanger and Gasser's[7] modification of this solution, may be employed. These colloidal solutions, if used early, can permanently raise arterial pressure. They do so by increasing the circulating volume of fluid. The corpuscles which are present are made to circulate more rapidly and thus to be employed more efficiently as oxygen carriers. There is no evidence that either the subcutaneous or intravenous injection of physiologic sodium chlorid solution has more than temporary value, if it has any beneficial effect at all.

SENSITIVENESS OF THE SHOCKED TO OPERATIVE PROCEDURES

In the earlier series of articles the sensitiveness of the shocked man to operative procedures was emphasized. Work done in cooperation with Lieutenant Cattell at the Surgical Research Laboratory at Dijon revealed the fact that not the operation but the etherization was the chief reason for the fall of pressure. If shock is already present in an animal, etherization until the wink reflex disappears may cause the pressure to fall 30 or 40 mm. of mercury (Fig. 4). On the other hand, the same degree of anesthesia may be induced by using nitrous oxid and oxygen in a ratio not exceeding 3 : 1 without any fall of blood pressure. There is little difficulty in bringing about satisfactory anesthesia or analgesia in shock cases with this ratio of the gases if there has been a previous dose of morphin. From suggestions which have been given above, it should be clear that morphin should not be given in such amount as greatly to retard respiration; and it should be clear also that at all stages deep anesthesia, or cyanosis or such rebreathing as would diminish the oxygen supply in the respired air, should be scrupulously avoided.